

Rounding of abrupt phase transitions in brain networks

Paula Villa Martín, Paolo Moretti, and Miguel A. Muñoz

Departamento de Electromagnetismo y Física de la Materia and Instituto Carlos I de Física Teórica y Computacional, Facultad de Ciencias, Universidad de Granada, 18071 Granada, Spain

Abstract. The observation of critical-like behavior in cortical networks represents a major step forward in elucidating how the brain manages information. Understanding the origin and functionality of critical-like dynamics, as well as their robustness, is a major challenge in contemporary neuroscience. Here, we present an extensive numerical study of a family of simple dynamic models, which describe activity propagation in brain networks through the integration of different neighboring spiking potentials, mimicking basic neural interactions. The requirement of signal integration may lead to discontinuous phase transitions in networks that are well described by the mean-field approximation, thus preventing the emergence of critical points in such systems. Here we show that criticality in the brain is instead robust, as a consequence of the hierarchical organization of the higher layers of cortical networks, which signals a departure from the mean-field paradigm. We show that, in finite-dimensional hierarchical networks, discontinuous phase transitions exhibit a rounding phenomenon and turn continuous for values of the topological dimension $D \leq 2$, due to the presence of structural or topological disorder. Our results may prove significant in explaining the observation of traits of critical behavior in large-scale measurements of brain activity.

1. Introduction

Experimental evidence of critical or quasi-critical behavior in brain networks was gathered over the past decade [1, 2, 3, 4, 5, 6, 7]. The discovery of scale-invariant avalanches of neural activity led to the conjecture that the brain might operate close to a critical point [1, 2]. It was argued that critical behavior might bear functional advantages; for instance, the divergence at criticality of quantities such as susceptibilities and correlation lengths could entail the ability of brain networks to coordinate system-wide activities and efficiently respond to a broad range of stimuli. A vast number of studies have since flowered, focusing on the numerical simulation of simple dynamical models that could recover phenomenologically the hallmarks of criticality observed in experiments [8, 9, 10, 11, 12]. In particular, it was noted that effective highly-simplified models of activity propagation –such as the contact process and the quiescent-excited-refractory-quiescent model– could provide valuable information on large-scale brain properties [13]. In these ideal models, an active “unit” or node –be it a neuron at a microscopic scale or a coarse-grained active region at a larger mesoscopic scale– can propagate its activity to neighboring units and/or become deactivated. Such simple dynamics –where activity propagation involves a single active node– lead generically to continuous phase transitions, with a critical point separating an active from a quiescent phase [14, 15, 16, 17]. Moreover, relatively simple modifications of these models implementing standard mechanisms of self-organized criticality lead to robust critical or quasi-critical behavior without the need of parameter fine tuning [18, 19, 20].

However, a closer look at real neural dynamics suggests that neural activity propagation may follow more complicated rules. In particular, individual neurons usually require to integrate up to hundreds of post-synaptic potentials before spiking themselves, as typically captured by integrate-and-fire models [21, 22, 23]. At mesoscopic scales, such requirement may be less stringent; however it is reasonable to consider that a few neighboring active units might be required to generate further activity: i.e. the dynamics follow a schematic rule of the type: $nA \rightarrow (n+m)A$, with $n > 1$ and m of the order of a few units, where each A stands for an active location or site [14, 15, 16, 17].

Such types of (n, m) -processes are well known in reaction-diffusion systems [14, 15, 16, 17], and they are often used in the modeling of neural dynamics. In particular, it is known that they lead to broad phases of sustained activity [8] and enhanced dynamic ranges [24, 25]. However, these processes are also well-known to lead to pattern formation (Turing patterns) and to discontinuous phase transitions between active and quiescent phases, with associated phase coexistence [26] and lacking critical points, in seeming contradiction with the observation of scale-invariant behavior in brain dynamics. In other words, the requirement of more than one source of activity to generate further activity leads to discontinuous phase transitions, separating two highly different active and quiescent phases, with no sign of criticality nor scale-invariance in between [14, 15, 16, 17].

Our goal here is to reconcile the need for signal integration at the neuron scale,

supposedly leading to discontinuous transitions, with the empirical observation of critical-like features, characteristic of continuous phase transitions. As we hope to convincingly argue, the key to this puzzling ambiguity lies in the topology of the underlying network of neural connections, which we will prove responsible for the generic rounding of discontinuous transitions. In a nutshell, and in analogy with what happens in problems of thermodynamic equilibrium at low dimensions, the existence of some form of structural disorder implies that potentially discontinuous transitions are rounded-off, thus making the system critical.

2. The underlying network of neural connections

In spite of the huge complexity that would be required to represent the detailed structure of the brain down to the single-neuron level, an effective coarse-grained description of neural contact patterns can be provided by a network –the connectome– whose nodes represent groups of neurons, such as cortical columns, and whose links represent the groups of fibers connecting them [9].

Studies employing different neuroimaging techniques have revealed that the Human Connectome, the current mapping of human brain connections, is organized in a hierarchical and modular fashion, in which local regions are clustered into large-scale moduli, which in turn form higher level structures and so on [27, 28, 29, 30, 31, 32]. The resulting hierarchical modular network (HMN) can be visualized as built-up from moduli of large internal neural connectivity, enclosed into higher-level sparser moduli, in a nested hierarchical fashion.

HMNs have been recently found to play a crucial role in neural dynamics. In particular, simple models of activity propagation were recently found to display Griffiths phases when running on top of HMNs [33], corroborating the experimental observation of extended critical regions in the human at its resting state [34]. Similarly, they were argued to extend the region of apparent criticality in self-organized models of neural activity and they were used to explain the ability of the brain to sustain activity over extended time windows [8, 9, 10, 11, 12, 33].

Here we shall use a simple structural model to build-up synthetic HMNs as follows: local densely connected moduli are used as building blocks; they are recursively grouped by establishing additional inter-moduli links in a level-dependent way, as exemplified in Figure 1. Further details of the construction methods can be found in Reference [33]. A crucial feature of HMNs is represented by their finite topological dimension D . The topological dimension of a network can be defined as follows: starting from a single node, the number of neighbors N_z reachable after z steps is computed for increasing z until the entire network is covered [35]. The network is finite dimensional with dimension D if $\langle N_z \rangle \sim z^D$, generalizing the familiar behavior of regular lattices. The topological dimension of a HMN can be tuned easily, by changing the average number α of links between pairs of modules at each hierarchical level (see Fig. 1 and [33]). Although brain moduli and columns may be densely connected, at larger mesoscopic and macroscopic

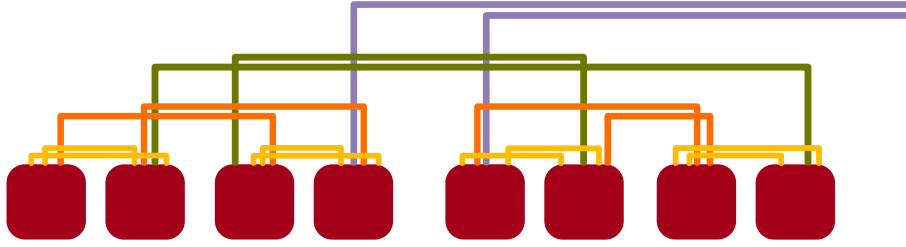


Figure 1. Sketch of the HMN construction method. Given a positive integer s , consider 2^s basal fully connected moduli of size M . At the lowest hierarchical level, moduli are linked pairwise into super-moduli by establishing a fixed number α of random unweighted and undirected links between the elements of each modulus ($\alpha = 2$ in figure). Newly formed blocks are then iteratively linked pairwise with the same α for a total of s iterations, until the network becomes connected. The resulting network has size $N = 2^s M$.

scales the hierarchical contact patterns become very sparse. At such scales, the effective network becomes finite dimensional [36, 33].

3. Continuous versus discontinuous transitions in the presence of disorder

It was recently conjectured that dynamical models of activity propagation characterized by discontinuous phase transitions at the mean-field level exhibit a rounding phenomenon in finite dimensional disordered systems, eventually leading to continuous phase transitions at dimension $D \leq 2$ [37]. Such behavior has been envisaged as the non-equilibrium analogue of the well-know Imry-Ma criterion, which states that –in the presence of quenched disorder– spontaneous symmetry breaking as well as first-order phase transitions are prohibited in equilibrium systems at $D \leq 2$ [38, 39, 40]. Analogously to quenched disorder in lattices, structural disorder is integral to HMNs as defined above and may thus be responsible for the rounding of discontinuous phase transitions in such systems [35]. In this light, we investigate how this form of topological disorder can potentially alter the order of phase transitions exhibited by simple (n, m) -models of neural activity, which would normally exhibit first-order phase transitions in networks well described by the mean-field approximation. In analogy with the Imry-Ma criterion, *a priori*, this effect should be expected to occur in networks with topological dimension D less than 2 [37].

4. Results

In what follows, we provide extensive numerical tests of the above conjecture, showing how the topological dimension of a disordered network can tune the nature of the dynamical phase transition, ultimately forcing $nA \rightarrow (n + m)A$ dynamics to exhibit continuous transitions for $D \leq 2$. To this end, we consider a prototypical model, in which we choose $n = 2$ and $m = 1$ (a $(2, 1)$ -process in our notation), whose Monte

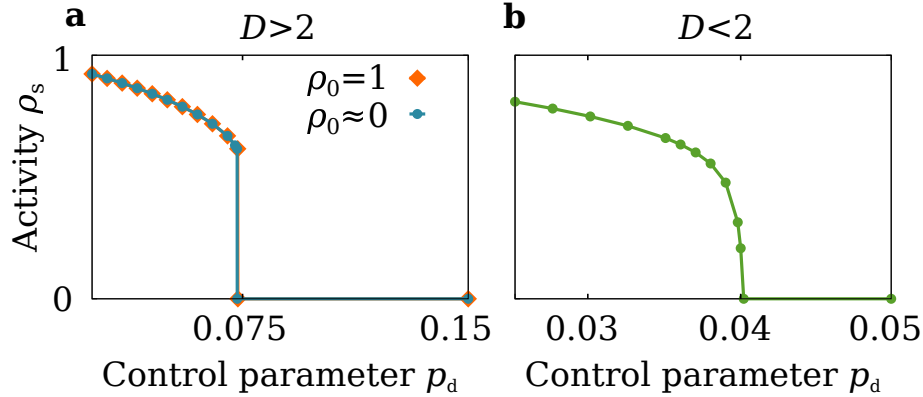


Figure 2. (Color online) Phase diagrams for topological dimension D above and below $D = 2$ respectively. In high dimension, the phenomenology of a discontinuous phase transition is recovered, in agreement with the mean-field prediction for the $(2, 1)$ -process. Below $D = 2$, the transition becomes continuous. A feeble appearance of hysteretic behavior is recorded in the $D > 2$ case, where different colors correspond to different initial conditions and spinodal points marking the transition are located at $p_{d,\text{thr}} \approx 0.0732(1)$ and $p_{d,\text{thr}} \approx 0.0734(1)$ for $\rho_0 = 0$ and $\rho_0 = 1$ respectively (not distinguishable in figure). Such dependence disappears for $D < 2$, in accordance with the hypothesis of a continuous phase transition, the critical point being located at $p_{d,\text{thr}} \approx 0.0402(1)$. Simulations are run on HMNs of size $N = 2^{17} = 131072$, partitioned into $s = 13$ hierarchical levels.

Carlo implementation is as follows: each of the N nodes of the network is endowed with a binary state variable $\sigma = 0, 1$, inactive or active, and $\rho(t)$ is the density of active nodes at time t ; i) at each time step an active node R_1 is selected and time is increased by $[N\rho(t)]^{-1}$; ii) with *death* probability p_d , R_1 is deactivated, while with complementary probability $1 - p_d$, a neighboring node R_2 is considered and one of the following actions is taken; iii) a) if R_2 is inactive, activity diffuses to R_2 , leaving R_1 ; iii) b) if R_2 is active, a new neighbor R_3 of R_1 is considered and, if inactive, it is activated with *birth* probability p_b . From a neurophysiological perspective, p_d encodes the exhaustion mechanism that accounts for spontaneous deactivation of neurons and neural regions, which proves essential in maintaining sustained activity bounded [8]. The integrated activation is tuned by p_b . A simple mean-field equation for this type of dynamics is

$$\dot{\rho}(t) = -p_d \rho(t) + (1 - p_d) p_b \rho^2(t) [1 - \rho(t)] \quad (1)$$

which exhibits a discontinuous phase transition (fold bifurcation) at $p_d = p_b / (4 + p_b)$ [26]. Notice that different choices of $n, m > 1$, which might account for enhanced realism in the physiological description of brain networks at the mesoscale, would not affect such behavior. On the other hand, a more detailed theoretical description of this system – taking explicitly into account the underlying network topology – would be provided by a quenched-mean-field approach [41, 42].

As a substrate on which the above dynamics run, we considered different HMNs, characterized by different topological dimensions D . In the rest of the paper we will

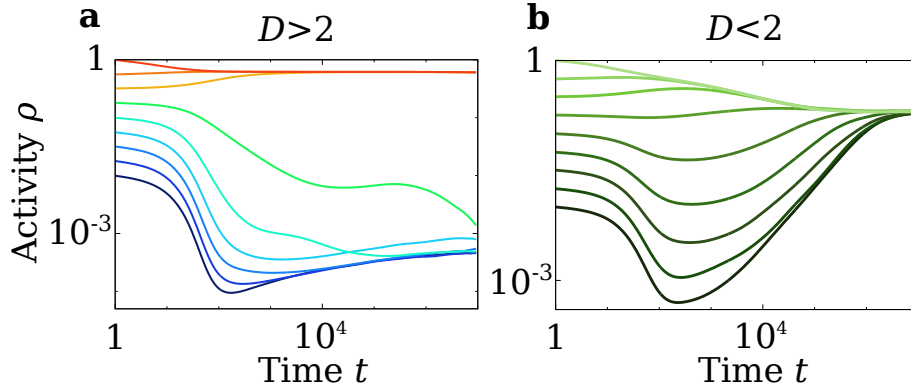


Figure 3. (Color online) Time evolution of the activity density ρ for different initial values ρ_0 (different colors), for dimension above and below $D = 2$ respectively. In both cases, the control parameter p_d is chosen at the threshold value. For high dimension, bistable behavior is recovered, as in standard first-order transitions, whereas no sign of bistability is encountered below $D = 2$. Notice however that both configuration converge very slowly to their expected behavior. In particular, in the $D > 2$ (discontinuous) case, large enough initial conditions lead to very long transients, which could be misinterpreted as continuous behavior for short simulation times. Such traits of quasi-critical states become stronger as $D = 2$ is approached from above, and corroborate the picture of a rounding phenomenon.

show results for HMN extracted from two ensembles \mathcal{N}_- and \mathcal{N}_+ , each with a fixed average dimension $D_{-,+}$ below and above the threshold value $D = 2$. In particular, we show results for $D_- \approx 1.6$ and $D_+ \approx 2.8$ (networks with such properties are obtained by choosing $\alpha = 1$ and $\alpha = 4$ respectively, in the HMN building-up process; see Figure 1).

We ran Monte Carlo simulations of the above $(2, 1)$ -process on such networks. Figure 2 shows the steady state value of the average activity density, as a function of the control parameter p_d , respectively below and above dimension $D = 2$, in spreading simulations starting both from localized active seeds ($\rho_0 \approx 0$) and from the homogeneously active state ($\rho_0 = 1$). While the discontinuity encountered above $D = 2$ is in agreement with the mean-field behavior for this type of dynamics, below $D = 2$ the dynamical phase transition is evidently continuous, confirming the conjecture of a low-dimensional rounding.

To provide further evidence of the radical difference in the transition nature, Figure 3 shows the time evolution of the average activity density ρ upon changing the initial activity ρ_0 , for both cases in Figure 2, each at the estimated threshold p_d . Above $D = 2$ clear signs of bistability emerge, signaling coexistence phenomena, which typically characterize discontinuous phase transitions. Below $D = 2$, however, the steady state does not depend on the initial condition anymore, as expected for a continuous transition, in which correlations become system-wide and coexistence is prohibited. In order to gain a deeper understanding of the rounding phenomenon, we can analyze the nature of the inactive (absorbing) phase in both cases. To this

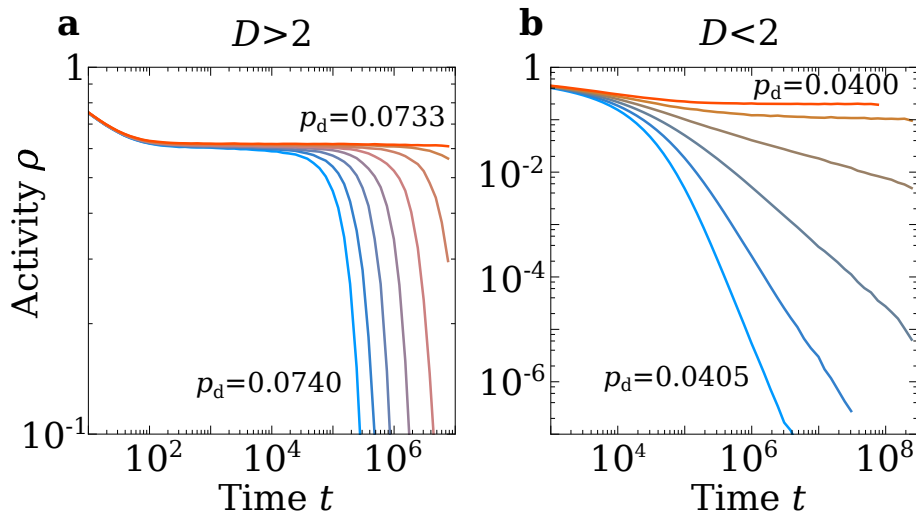


Figure 4. (Color online) Time evolution of the activity density ρ for different values of the control parameter p_d (different colors) below the dynamic threshold, for dimension above and below $D = 2$ respectively. For high dimension, the transition between the inactive and the active state is abrupt, with no signs of criticality. Below $D = 2$, a Griffiths phase emerges for $p_d > p_{d,thr}$, characterized by generic power-law relaxation and critical-like behavior.

end, let us consider simulations starting from a homogeneous $\rho_0 = 1$ state. Time evolution of ρ is shown in Figure 4, for different values of p_d in the inactive phase. As usual, above $D = 2$ results for p_d point to an abrupt change in behavior at the dynamic threshold $p_{d,thr}$, below which activity dies off exponentially fast as soon as a large enough fluctuation breaks the coexistence of active and inactive islands. At $p_{d,thr}$, such coexistence becomes stable in the large- N limit and the phenomenology of a discontinuous phase transition is recovered. For dimensions below $D = 2$, instead, the system displays a Griffiths phase[43, 44]: the average activity density decays as power laws with continuously varying exponents as a function of the control parameter p_d . A critical point, characterized by activated scaling logarithmic time decay ($p_{d,thr} \approx 0.0402(1)$ in Fig.4) separates the Griffiths phase from the active phase, marking the recovery of critical behavior at low dimension. Griffiths phases are a manifestation of rare-region effects: islands of localized activity are able to remain active for long times. Their relevance for both complex networks [35] and brain networks [33] has been recently discussed in the literature. Activity propagation models without signal integration yield such behavior in HMNs as they naturally lead to continuous phase transitions regardless of dimensionality constraints [33]. Remarkably, the $(2, 1)$ -model dynamics at low dimensions recovers here those fingerprints of criticality, in spite of being typically associated with discontinuous transitions at mean field. Interestingly, upon approaching the threshold dimension from above, $D \rightarrow 2^+$, the discontinuous nature of the transition is rounded: although coexistence is genuinely recovered at very large times, activity is able to self-sustain even in the absorbing state for times

potentially longer than any observation window.

5. Discussion and Conclusions

A recent study showed that the $(2, 1)$ -process adopted here as a paradigm for first-order phase transitions may show tricritical behavior in certain families of ordered fractal lattices of dimension $1 < D < 2$ [45]. Such a finding implies that for each family of ordered fractals, there exist a “critical” dimension $1 < D_c < 2$, below which the transition is continuous, recovering the known behavior of one dimensional chains, and above which the transition becomes discontinuous, anticipating the behavior of pure two-dimensional lattices. In our study we have introduced disorder in the topology and shown that finite-dimensional disordered hierarchical modular networks of relevance in neuroscience *always* display continuous phase transitions for $D < 2$. In fact even square lattices ($D = 2$) exhibit this behavior provided that disorder is introduced, specifically in the form of quenched impurities [37]. Such results corroborate the conjecture that, due to disorder, non-equilibrium systems with absorbing states do not sustain first-order dynamical phase transitions for *any* $D \leq 2$. We provided further evidence for this claim, proving its validity for HMNs, and we focused on the relevance that such result may have for neuroscience. Brain activity is known to exhibit critical-like behavior, which would suggest its ability to sit constantly in the vicinity of a continuous transition. We have shown that even if realistic dynamic models lead to first-order phase transitions in the mean-field approximation, in low-dimensional disordered systems such transitions are rounded. A natural question arises whether the brain actually is a low-dimensional network, provided that each single neuron may have up to thousands of neighbors. The solution to this apparent contradiction comes from the hierarchical organization of brain connections. At the lowest scales, neurons are grouped in well connected moduli which act as small worlds of diverging topological dimension. At such scales, integrate-and-fire dynamics naturally trigger coexistence and local discontinuous activations of moduli. At the largest scales, however, inter-moduli connectivity is very sparse in order to maintain the volume of white-fiber matter bounded [9], allowing only for weak small-world effects [36]. Such connectivity patterns become finite dimensional, and discontinuous phase transitions are prohibited. Notice that the $D = 2$ bound should not be read strictly in real systems. We found that significant traits of quasi-critical behavior appear even above $D = 2$, suggesting a gradual rounding phenomenon. Such systems will theoretically show discontinuous transitions for large enough times, yet they are able to sustain anomalous activity for typical time windows of experimental observations. More detailed and realistic models of neural dynamics could be provided. While the behavior presented here is conserved for different choices of the parameters (n, m) , realistic models supposedly include ingredients such as refractory times, explicit time integration, inhibition and dependence on synapse directedness. While such details are of primary importance in correctly describing physiological aspects of brain activity, we believe that our simple approach has the advantage of focusing on the large-scale

topology of the Human Connectome [27, 31], in order to provide insight about its large-scale behavior. An understanding of low-level synaptic activity requires realistic neuron models and remains a formidable task.

In conclusion, we have studied the properties of a family of dynamic models of relevance in the description of neural activity in the presence of signal integration. Although signal integration may be responsible for the emergence of first-order phase transitions in generic networks, we have shown that phase transitions are rounded in finite dimensional hierarchical networks, eventually turning continuous for $D \leq 2$. Such finding is relevant in explaining the observation of critical behavior in the brain at large scales, in spite of the high degree of signal integration required to fire neuron activity at small scale.

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